BIBLIOGRAPHY.

1. Batten: Ataxia in Childhood, Brain, 1905, xxviii, p. 484.
2. Batten: Congenital Cerebellar Ataxia (Cerebellar Diplegia). Clin. Jour., 1903, xxii, p. 81.
3. Batten and von Wyss: The Atonic Form of Cerebral Diplegia, British Jour. Child. Dis., 1915, xii, p. 65.
4. Brissaud: Maladie de Little, Semaine méd., 1894, xiv, p. 89.
5. Clark, L. P.: Infantile Cerebrocerebellar Diplegia of Flaccid Atonic-Astasic Type, Am. Jour. Dis. Child., 1913, p. 425.
6. Cushing, H.: Concerning Surgical Interference for Intracranial Hemorrhage of the Newborn, Am. Jour. Med. Sc., 1905, cxxx, p. 563.
7. Förster: Der Atonische-Astatische Typus der Infantillen Cerebral Lähmung, Deutsch. Arch. f. klin. Med., 1909, xcviii, H. 1 and 3, S. 216.
8. Gowers: Infantile Meningeal Hemorrhage, Manual of Nervous Diseases, i, p. 413.
9. Gowers: Birth Palsies, Lancet, 1888, ii, p. 707.
10. Holmes and Sargent: Injuries of the Superior Longitudinal Sinus, British Med. Jour., 1915, xxiii, p. 493.
11. Hunt: Thrombosis of the Cerebral Sinuses and Veins as Complication of the Puerperium, Bull. Lying-in Hospital, May, 1917, xi, No. 2, p. 73.
12. Hunt: Progressive Atrophy of the Globus Pallidus (Primary Atrophy of the Pallidal System), a System Disease of the Paralysis Agitans Type, Characterized by Atrophy of the Motor Cells of the Corpus Striatum. A Contribution to the Functions of the Corpus Striatum, Brain, 1917, xl, Part I, p. 58.
13. Kundrat: Ueber die Intermeningealer Blutungen Neugeborenen, Wien. klin. Wchnschr., 1890, iii, p. 887.
14. Koenig: Beiträge zur Klinik der Cerebralen Kinder Lahmungenen, Deutsch. Zeitschr. f. Nervenh., 1901, xv, S. 479.
15. Little: On the Influence of Abnormal Parturition, Difficult Labor, Premature Birth and Asphyxia Neonatorum on the Mental and Physical Condition of the Child, Especially in Relation to Deformities, Tr. Obst. Soc. London, 1862, iii, p. 293.
16. MacNutt: Apoplexia Neonatorum, Am. Jour. Obst., 1885, xviii, p. 73.
MacNutt: Double Infantile Spastic Hemiplegia, Am. Jour. Med. Sc., January, 1885, cxiv.
17. Marfan: La Bigidite Spasmodique Congenitale (Maladie de Little), Presse Méd., 1894, ii, p. 139.
18. Oliver: The Cerebral Birth Palsies, British Med. Jour., 1893, i, p. 733.
19. Railton: Birth Palsy, British Med. Jour., 1892, xxvii, p. 441.
20. Schultze, Fr.: Haemato-myelie und Oblongata Blutung bei Dystokien, Deutsch. Zeitschr. f. Nervenh., 1895, viii, p. 1.
21. Spencer, H. R.: On Visceral Hemorrhages in Stillborn Children, Tr. Obst. Soc. London, 1891, xxxiii, p. 203.
22. Thomas, André and Jumentie: Rev. Neur., 1914, i, p. 566 (Atonic Case).
23. Van Gehuchten: Faisceau Pyramidal et Maladie de Little, Jour. de Neur. et d'Hyp., 1896, i, p. 266.
24. Von Monokow: Die Lokalisation im Grosshirn (Localization der sensibilitat im Kortex), S. 300.
25. Vogt, H.: Cerebral Diplegia, Lewandowsky's Handbuch der Neur., vol. ii.
26. Voisin et de Lepinay: Syndrome Cerebelleux congenitaux, Rev. Neur., 1907, xv, p. 395.

ANGINA ABDOMINIS.[1]

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The term angina abdominis was introduced by Baccelli to describe painful seizures in the abdomen due to aneurysmal for-

[1] Read before the American Gastro-enterological Association.

mations in the vessels of the celiac axis, or due to arteriosclerosis of these same vessels. Another name for the same condition has been offered by Huchard, angina pectoris pseudogastralgique. Inasmuch as the name invented by Baccelli has been accepted and used by recent writers, notably Pal, Brunton and Williams, Minella, Breitmann and others, it is the term which should be used in the future for this disease.

Case History. The patient, a well-preserved woman, aged eighty-two years, apart from trivial illnesses of but short duration, has enjoyed good health for the greater part of her life. Twelve years ago she suffered with intense pain in the stomach, which was diagnosed at this time carcinoma ventriculi. In view of the present state of health the diagnosis is subject to question. Disregarding this attack, which resembled the seizure recently experienced, the patient rejoiced in the best of health and spirits until the early part of September, 1916. Following hard physical work connected with housecleaning, a task which no appeal to regard her years could make her depute to others, she was stricken with violent and intense pain in the umbilical region, which was accompanied by great anxiety, nervousness, restlessness and attempts at expulsion of gas per rectum and ex ore. These attacks having no relation to meals were repeated frequently, medication and diet proving unavailing, since September. She was seen by a surgeon in November, 1916, as gall-stones were suspected, but the diagnosis was not confirmed. Operation was disadvised, but no satisfactory conclusion was reached concerning the nature of the attacks. The surgeon's examination, which was most thorough, failed to reveal any organic lesion other than the arteriosclerosis.

The patient was counselled to remain in bed, to partake of nothing except milk, and was given a mixture of hydrochloric acid and pepsin, with occasional doses of sodium bicarbonate and nutmeg. For three days she was so much better that her physician permitted her to get out of bed, and again with utter disregard for her years, she cleaned a closet and laid unassisted a heavy carpet (November 23), being alone in the house at this time. Following this exertion she was seized with an excruciating pain in the umbilical region, with restlessness, extreme nervousness, attempts at expulsion of gas, no abdominal distention, no passage of urine or feces, some nausea, but no vomiting. The following morning this attack was duplicated in all characteristics except that the pain was less formidable. I saw the patient in company with the attending physician approximately about four hours after this second, and to date, last attack. The seizure on this day had been of short duration, as were all the others, that is to say, the acme of the frightful pain was sustained for only a few minutes, but intense abdominal soreness followed, and was present when I saw her, and lasted for a period of about twenty-four hours.

I found the patient (November 24) to be a remarkably well-preserved old lady, of frail build. The pulses were equal, regular, normal in rate, but the vessels were hard and ensily palpable. The heart examination revealed a faint systolic murmur at the hase, which was interpreted as being caused hy aortic roughening.

The stomaeh was slightly dilated, there was n splash (milk had been ingested but a short time before) and some diastasis of the recti. The abdomen was very sensitive to pressure, particularly in the umbilical region, corresponding to a eireular area about 6 inches in diameter, having the umbilicus as its center. There was marked throbbing of the nbdominal aorta, no evidence of any enlargement of the vessels, and no murmurs. The reflexes were normal nnd the lower legs were slightly edematous. A reetal examination made by the surgeon five days previously, and being reported negative, was not repeated. The urine has contained albumin, bnt no sugar or casts. The blood-pressure was not taken at this time, as I had neglected tn take my instrument, having been told it was essentially a stomach case. My neglect in this respect I have since regretted, ns my request to the attending physician for n subsequent report of the blood-pressnre was overlooked. However, four months later the pressure was found to be 130–60.

I believed we were confronted with an almost typical case of nnginn nbdominis, and although skeptical as to the correctness of this view, the physician consented to give my therapeutic recommendations n trial. To the patient was explained what we believed to be the cause of her distressing attacks, nnd we gained her support in the view tbnt physical overexertion or nervous strain, or both, were predisposing fnctors in calling an attnck into being. Therefnre the recommendatinn tn rest during the day, and partieularly a half-hour's rest on the right side, with a hot-water bottle to her nbdomen following her meals, was readily agreed to. She was permitted to eat nnything sparingly at the regular hours for meals and a glass of milk with a biscuit between meals. Only meat was restricted. Nitroglycerin, gr. $\frac{1}{100}$, was given every three hours, and potnssium iodide, gr. x, three times a day. Pearls of nnyl nitrite were left in the event of a recurrence of the pain.

Since November 24 the patient experienced the beginning of an attack, but the attack was cut short by the prompt use of amyl nitrite, and since that date there have been no further attacks. Potassium iodide and nitroglycerin have long since been discontinued; the patient is active in Red Cross work, and seems to be enjoying the best of health, the fear of cancer having, to her great relief, been permnnently dispelled from her thoughts.

The symptoms as disclosed by the history of this patient seem almost typical of what has been described by others ns nngina nbdominis, and a further diseussion of the symptomatology of n classical case would seem unnecessary. Permit me to recall the

cardinal facts. The main feature of this woman's illness was the sudden agonizing pain which was directly associated with physical overexertion or nervous shock, thus in a way resembling the onset of angina pectoris. There is a prevalent notion that all attacks of angina pectoris are determined by emotional or physical effort, yet from Heberden himself we learn that attacks may occur during sleep and often during rest. Diderot records an attack which aroused him from sleep (Allbutt). Nevertheless, many cases of angina pectoris are precipitated by exertion, and in this patient of mine effort seems to have been the exciting factor. The pain is described by the patient as being horribly severe, and after the first seizure the dread of another and the realization that others were to occur, since the attacks were repeated, made her life one of continuous apprehension.

Accompanying the pain there were efforts at expulsion of gas by mouth and rectum, and when belching could be effected there seems to have been some slight relief. The bowels were slightly constipated. There was slight nausea but no vomiting.

The situation of the pain was about the umbilical region and radiated to the back, and following the seizure there was great sensitiveness on slight pressure about the umbilical region for an area 6 inches in diameter.

The diagnosis should not be difficult, and to my mind the main support of the correct diagnosis should be based on the frightfulness of the pain, the short duration of the same, the occurrence of the pain in arteriosclerotic individuals, usually advanced in years, in whom, apart from the arteriosclerosis, there can be found no appreciable sign of disease. Men are said to be more frequently affected than women.

The differential diagnosis must, in some cases, be made between abdominal angina and other diseases associated with gastric crises. Here it may be stated that Allbutt believes that epigastric angina is but a somewhat aberrant form of ordinary angina, and due, broadly speaking, to disease of some part of the thoracic aorta; but in angina "more definitely abdominal," he thinks it may be due to disease of the lower portion of the aorta or in other large vessels there. Osler is disposed to discredit the diagnosis, regarding abdominal angina as but the abdominal symptoms of angina pectoris. In this patient of mine, despite her years, no symptom of any cardiac disease had been complained of. There were no transient seizures without pain in which "there was a general suspension of the minor operations of nature for three or four seconds," called angina sina dolore.

Abdominal aneurysm should not be a matter of much moment in the differential diagnosis, as the pain is never anginoid, but rather boring in character, and situate in the left abdominal region. (Of 179 cases reported by Nixon none had anginoid pains.)

Tabes dorsalis should be readily distinguished, as the reflexes point the way to the true diagnosis, unless, unhappily, the patient is a sufferer from both tabes and from angina abdominis. The pain in tabes is intercostal rather than abdominal.

Closure of the mesenteric vessels I mention as an improbable differential diagnostic difficulty, on account of its extreme infrequency and because of the impossibility of recognizing the condition, unless one has been the observer of a previous case. Closure of the mesenteric vessels may take place in either the veins or arteries as a result of thrombosis or embolism. For thrombosis, arteriosclerosis itself is an underlying predisposing cause; for embolism valvulitis is the contributing factor. The onset of mesenteric closure is sudden and leads rapidly with great pain to collapse. Bloody vomitus is seen, and the stools, which contain blood, may be either obstipated or diarrheic. The abdomen becomes distended, painful and resistant to touch, with dulness in the dependent portions of the abdomen. The temperature is normal, pulse is rapid and soft and the patient gives the impression of one suffering with severe peritonitis or intestinal obstruction. In no case should abdominal angina be mistaken for mesenteric closure.

I should prefer to leave the question of pathology undiscussed, as but few cases have been studied satisfactorily from the clinical and pathological sides. Various suppositions have been suggested, but "clinical and pathological precision has yet to contribute to a better analysis of these phenomena." (Allbutt.)

So far as therapy is concerned, rest is an important measure, and avoidance of physical or nervous strain is to be recommended. Diet is to be that generally recommended for individuals of advanced years. Massage and faradization of the abdomen have been advised, and the homely hot-water bottle is not to be despised. Potassium iodide, sodium nitrite, sodiotheobromin, salicylate, nitroglycerin, amyl nitrite may each have its place, alone or in careful combination. During an attack if amyl nitrite fails, morphin, hypodermically, may be employed.

THE RELATION OF LUPUS ERYTHEMATOSUS DISCOIDES TO TUBERCULOUS INFECTION.

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The etiology of lupus erythematosus has been one of the obscure problems of dermatology ever since the first description of the dis-